Environmental Tobacco Smoke and Sudden Infant Death Syndrome: A Review

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Environmental tobacco smoke (ETS), containing the developmental neurotoxicant, nicotine, is a prevalent component of indoor air pollution. Despite a strong association with active maternal smoking and sudden infant death syndrome (SIDS), information on the risk of SIDS due to prenatal and postnatal ETS exposure is relatively inconsistent. This literature review begins with a discussion and critique of existing epidemiologic data pertaining to ETS and SIDS. It then explores the biological plausibility of this association, with comparison of the known association between active maternal smoking and SIDS, by examining metabolic and placental transfer issues associated with nicotine plus the biological responses and mechanisms that may follow exposure to nicotine. Evidence indicates that prenatal and postnatal exposures to nicotine do occur from ETS exposure, but that the level of exposure is often substantially less than levels induced by active maternal smoking. Nicotine also has the capacity to concentrate in the fetus, regardless of exposure source. Experimental animal studies show that various doses of nicotine are capable of affecting a neonate's response to hypoxic conditions, a process thought to be related to SIDS outcomes. Mechanisms contributing to deficient hypoxia response include the ability of nicotine to act as a cholinergic stimulant through nicotinic acetylcholine receptor (nAChR) binding. The need for future research to investigate nicotine exposure and effects from non-maternal tobacco smoke sources in mid- to late gestation is emphasized, along with a need to discourage smoking around both pregnant women and infants.

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